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A modelling approach to vaccination and contraception programmes for rabies control in fox populations

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In a previous study, three of the authors designed a one-dimensional model to simulate the propagation of rabies within a growing fox population; the influence of various parameters on the epidemic model was studied, including oral-vaccination programmes. In this work, a two-dimensional model of a fox population having either an exponential or a logistic growth pattern was considered. Using numerical simulations, the efficiencies of two prophylactic methods (fox contraception and vaccination against rabies) were assessed, used either separately or jointly. It was concluded that far lower rates of administration are necessary to eradicate rabies, and that the undesirable side-effects of each programme disappear, when both are used together.

Keywords: discrete modelling; rabies; foxes; oral vaccination; contraception

1. INTRODUCTION

Fox rabies is a major veterinary public-health problem in several countries of the world (Blancou *et al.* 1991). Oral vaccination of foxes carried out by distribution of vaccine baits has had a clear impact on the prevalence of the virus in Western Europe (Stöhr & Meslin 1997; Pastoret & Brochier 1999).

Data from fox-hunting records indicate that the European fox population tends to increase (Artois 1997). This has been observed in both rabies-free (Great Britain, Tapper 1992; J.-A. Reynolds, personal communication) and rabies-infected areas (Belgium, De Combrugghe 1994; Germany, Müller 1995). This does not mean that fox populations are not regulated over the long term, but simply that over the short term the population income–outcome ratio is not balanced for unknown reasons (increase of resources and/or decrease in mortality). Whatever the cause of fox-population increase, it could eventually impede the success of further oral-vaccination campaigns when the number of non-immunized foxes becomes high enough to carry on the infection (Breitenmoser *et al.* 1995; Vuillaume *et al.* 1997). A sufficient level of culling to achieve a sustainable control of the population is difficult to obtain if the rabies threshold density is much lower than that of the population carrying capacity (Anderson *et al.* 1981). The combination of culling and vaccination is still a matter of debate (Smith 1995; Barlow 1996). A promising solution could be the limitation of recruitment of healthy foxes through fertility control. Increasing efforts have been focused on this technique for red fox predation control in Australia (Bradley 1994).

In a previous model, vaccination by the oral route alone was examined as a way of controlling rabies in high fox-population density areas (Artois *et al.* 1997). This model emphasized that a vaccination rate lower than 70% will allow the epidemic to persist, a figure already described by Smith (1995). In this study, the focus was on fertility control through the use of baits filled with a contraceptive vaccine in conjunction with a rabies vaccine as a possible method of controlling rabies when vaccination alone is not sufficient for disease eradication (Smith 1995).

2. DESCRIPTION OF THE MODEL

The present model was based on the one-dimensional discrete deterministic model of Artois *et al.* (1997). The fox population has been structured in space (a two-dimensional model in this paper with N home ranges), in age (young and adult, i.e. dispersing foxes or residents one year old and more), in sex (female and male) and in disease state (healthy, exposed and vaccinated). This gave 12 classes of foxes per cell through which rabies propagated (figure 1). The density of healthy young females in cell n at time t has been denoted by $\mathrm{HYF}(n,t)$, with analogous notations for the 11 other fox classes.

The time-step, $\Delta t = 10$ days, chosen in the simulations is longer than the life expectancy of clinically ill individuals (1–4 days) (Blancou *et al.* 1991). Thus no specific class of infectious individuals has been considered. Instead, the number of infectious individuals in the time interval from t to $t+\Delta t$ is proportional to the number of exposed individuals; the proportionality coefficient $\sigma(t)$ is the inverse of the latency period.

(a) *Demography*

As a further contrast with Artois *et al.* (1997), this paper simulates the demography of the fox population as either exponentially increasing or density dependent.

In a density-dependent fox population the natural mortality is different for young foxes and adult ones,

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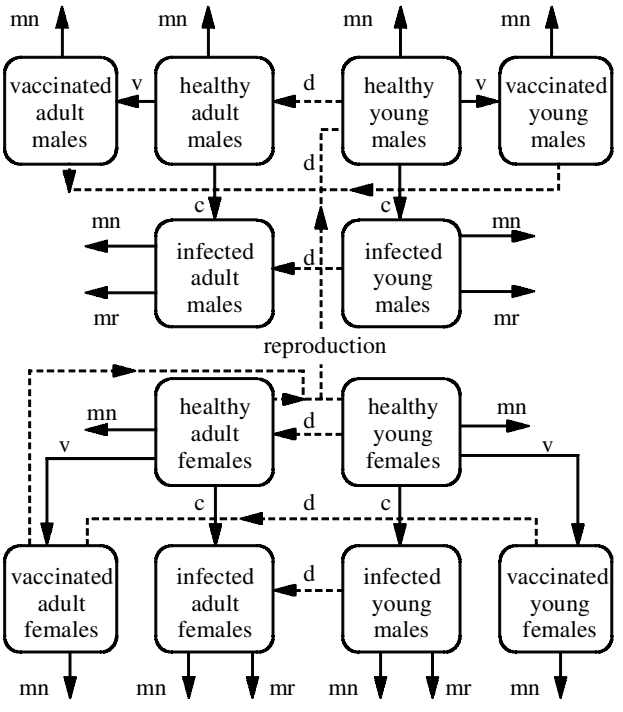

Figure 1. Interaction between the 12 classes of foxes. mn, natural mortality; mr, mortality induced by rabies; v, vaccination; d, dispersal; c, contamination.

according to season. Survival is therefore also density dependent: for adult female foxes it has been determined as

$$\mathrm{saf}(t) = \hat{\mathrm{saf}}(t) \times \frac{1}{1 + \delta(n,t)P(n,t)}; \quad (1)$$

where $\hat{\mathrm{saf}}(t)$ is the natural survival rate of adult female foxes, $P(n,t)$ is the total density of foxes living at time t in home range n and $\delta(n,t)$ is a non-negative parameter. When $\delta = 0$ there is no density dependence, while a positive δ will yield a logistic effect. Similar formulae have been used for other age and sex classes. In simulations, δ is a constant, numerically evaluated to supply an average fox density of $0.01\,\mathrm{ha}^{-1}$ (Artois 1989). Only healthy and vaccinated females were able to reproduce as incubation period was shorter than gestation and weaning duration; hence infected cubs had no chance of survival. The density of healthy young females in cell n is

$$\mathrm{HYF}(n,t+\Delta t) = b(t) \times \mathrm{saf}(t) \times \mathrm{HAF}(n,t), \quad (2)$$

where the birth function, $b(t)$, satisfies $b(t) = b_0$ on 1 April and $b(t) = 0$ otherwise (Artois 1989), with $2b_0$ being the average number of cubs per litter per female and b_0 referred to as the half birth rate.

(b) *Two-dimensional spatial structure and dispersion*

A rectangular domain is subdivided into cells having a hexagonal shape, each cell corresponding to the size of an average fox's home range. Cells have been numbered from 1 to N; the hexagon lying at the intercept of line i and column j is numbered

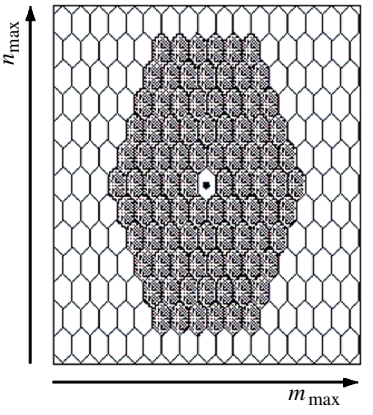

Figure 2. Structure of the two-dimensional domain. The shaded area represents the set of cells that a young fox living in cell n can reach through dispersal.

$$n = (i-1) \times n_{\max} + j, \quad (3)$$

where $n_{\max}$ is the maximal number of cells on a line (figure 2).

Conversely, the location (i,j) on the grid of a hexagon having number n could quickly be found from

$$i = I\left[\frac{n-1}{n_{\max}}\right] + 1, \; I[r] \text{ is the integer part of the real number } r, \quad (4)$$

$$j = n - (i-1) \times n_{\max}. \quad (5)$$

During the dispersal process, young foxes leave their parental home range to become territorial. In our model, we assumed that a young fox disperses one way along a straight path and crosses at most L home ranges before settling down (Lloyd 1980; Macdonald & Bacon 1982; Trewhella *et al.* 1988). Thus it can reach $1 + 3L(L+1)$ different cells (figure 2). In this model $D(n,L)$ was defined as the set of cells that a young fox living in cell n can reach through dispersal and as the set of cells from which a young fox arriving in cell n started from. Finally, the radial distance between two cells was determined through a simple algorithm.

The probability of a young fox settling in a given home range was assumed to depend only on the number of cells it crossed, i.e. the radial distance between the end-points of its path. As a model we took a linearly decreasing function of the distance travelled: the probability of reaching a cell located at a radial distance d is

$$\Psi(d) = \frac{\rho \times [(L+1) - d]}{6 \times d}, \text{ with } \rho = \frac{2}{(L+1)(L+2)}, \text{ for } d = 1, \ldots. \quad (6)$$

As an example, in a rabies-free situation, the densities of healthy young and adult females are given by the following equations:

$$\mathrm{HYF}(n,t+\Delta t) = (1 - F(t)) \times \mathrm{syf}(t) \times \mathrm{HYF}(n,t), \quad (7)$$

$$\mathrm{HAF}(n,t+\Delta t) = F(t) \times \mathrm{hydf}(n,t) + \mathrm{saf}(t) \times \mathrm{HAF}(n,t), \quad (8)$$

where $F(t)$ is the proportion of young foxes that disperse, $\mathrm{syf}(t)$ is the survival rate of young females, $\mathrm{saf}(t)$ is the survival rate of adult females and $\mathrm{hydf}(n,t)$ is the number of healthy young females that arrive in cell n.

At time t

$$\mathrm{hydf}(n,t) = \sum_{k\in D(n,1)} \varphi(k,n) \times \mathrm{syf}(t) \times \mathrm{HYF}(k,t), \quad (9)$$

where $\varphi(k,n)$ is the probability of a fox located in cell k coming into cell n at a radial distance d from cell k:

$$\varphi(k,n) = \psi(d). \quad (10)$$

As in the one-dimensional model, there was a problem for young foxes leaving a home range close to the boundary of the domain. Here we considered that young foxes that would have left the domain through dispersal remained in their parental territory, therefore no fox crossed the boundary. Under this assumption, numerical simulations show that in a rabies-free situation, the global dynamics is that of a large isolated population. Numerical simulations in a disease-free environment show no density increase at the edges. Since the aim of this paper was to compare the efficiency of different control strategies within the centre of the domain, the edge effect at the boundary could be neglected.

(c) *Transmission*

Transmission of rabies occurs through bites and licking, as the rabies virus is transmitted via the saliva (Blancou *et al.* 1991). It is thought that rabies can be propagated by two modes:

(i) Outside the dispersal of juveniles, between foxes living in the same or adjacent home ranges (Artois 1989). For a given cell, n, these home ranges have numbers

$$v_j(n) = n - n_{\max},\ n - n_{\max} + 1,\ n - 1,\ n,\ n + 1,\ n + n_{\max},\ n + n_{\max} + 1, \quad (11)$$

the density of infected young females being determined as

$$\mathrm{IYF}(n,t+\Delta t) = \mathrm{syf}(t) \times \mathrm{IYF}(n,t) + \beta_j(t) \times (\mathrm{syf}(t) \times \mathrm{HYF}(n,t)) \times I(n,t); \quad (12)$$

where $\beta_j(t)$ is the transmission rate from an infectious fox to a healthy young fox at time t, and $I(n,t)$ is the number of infectious foxes living in cell n and in the six surrounding cells determined as

$$I(n,t) = \sum_{v_j(n)} \sigma(t) \times [\mathrm{IYF}(v_j,t) + \mathrm{IAF}(v_j,t) + \mathrm{IYM}(v_j,t) + \mathrm{IAM}(v_j,t)]. \quad (13)$$

As indicated earlier $\sigma(t)$ is the inverse of the latency period.

(ii) During dispersal (October and November) (Artois 1989), infected young individuals carry the infection further than one home range (see figure 2). The density $\mathrm{IAF}(n,t+\Delta t)$ of infected adult females in cell n at time $t+\Delta t$ is given by

Table 1. *Data used in simulations*

studied area	$n_{\max} = 61$; $N = n_{\max} \times m_{\max} = 3721$
dispersal distance	$l = 5$, or 91 reachable home ranges
survival rate	
(Artois *et al.* 1997)	adult young
summer	0.99 0.97
winter	0.98 0.98
latency period	
(Blancou *et al.* 1991)	21 days or $\sigma = 0.48$
birth rate	$b(t) = b_0$ on 1 April; $b(t) = 0$ otherwise
transmission rate	$\beta(t) = \beta_a(t) = \beta$

$$\mathrm{saf}(t) \times \mathrm{IAF}(n,t) + \beta_j(t) + (\mathrm{syf}(t) \times \mathrm{HAF}(t)) \times I(n,t) + F(t) \times \mathrm{iydf}(n,t), \quad (14)$$

where $\mathrm{iydf}(n,t)$ is the number of infectious young females that could arrive in cell n at time t:

$$\mathrm{iydf}(n,t) = \sigma(t) \sum_{k\in D(n,l)} \varphi(k,n) \times \mathrm{syf}(t) \times \mathrm{IYF}(k,t). \quad (15)$$

(d) *Vaccination and sterilization programmes*

Two vaccination campaigns per year were simulated in our model (Aubert 1995): one in spring to target adult animals, and one in autumn to target all age classes. The number of healthy and vaccinated young females in a cell n at time $t+\Delta t$ has been determined as

$$\mathrm{HYF}(n,t+\Delta t) = (1 - \mathrm{vay}(t)) \times \mathrm{syf}(n,t) \times \mathrm{HYF}(n,t), \quad (16)$$

$$\mathrm{VYF}(n,t+\Delta t) = \mathrm{vay}(t) \times \mathrm{syf}(n,t) \times \mathrm{HYF}(n,t), \quad (17)$$

where $\mathrm{vay}(t)$ is the vaccination rate at time t.

In fertility-control campaigns, each autumn only females are concerned and contraception is only effective during one breeding season. To take contraception into account the equations of the previous model were modified: the number of healthy young females, or males, in a cell n at time $t+\Delta t$ was determined as

$$\mathrm{HYF}(n,t+\Delta t) = \mathrm{HYM}(n,t+\Delta t) = b(t) \times (1 - \mathrm{st}(t)) \times \mathrm{HAF}(n,t) \times \mathrm{saf}(n,t), \quad (18)$$

where $\mathrm{st}(t)$ is the sterilization rate at time t.

3. SIMULATION RESULTS

Numerical simulations were performed on a workstation using a Fortran 77 code on the data shown in table 1.

Various numerical values of the birth and transmission rates have been used. More precisely values for β and b_0 were determined that were consistent with either an endemic state or a disease-free state. The efficiency of fox contraception, dependent on or independent of vaccination against rabies was then easier to analyse.

In Artois *et al.* (1997) birth rate, b_0, was 2.5. Here b_0 varied from 1.02 (see § 3(a)) to a maximum of 3.5, which corresponded to seven cubs per litter per female, with a balanced sex ratio (Artois 1989).

In Garnerin *et al.* (1986) and Artois *et al.* (1997) transmission rate, β, was 0.18. Here β varied from 0.04 to a maximum of 0.20 (see § 3(b)).

Table 2. *Malthusian parameters for different birth rates*

birth rate	Malthusian parameter
1.02	0.0001
1.5	0.005
2.0	0.0095
2.5	0.0133
3.0	0.0166
3.5	0.0182

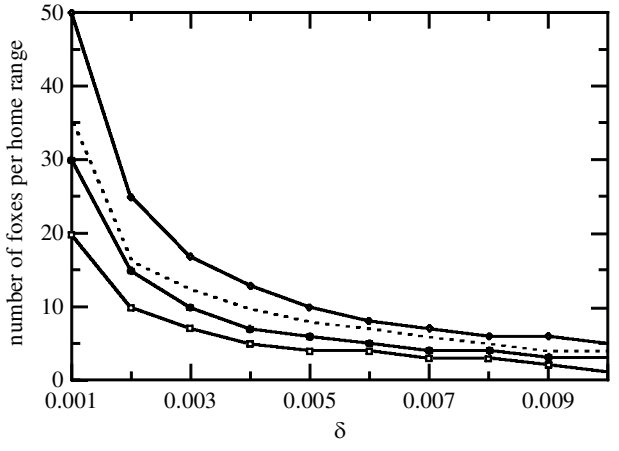

Figure 3. Number of young and adult foxes per cell as a function of the density-dependent parameter δ for $b_0 = 3.5$ (top curve), 3, 2.5 and 2 (bottom curve).

Actually, β is unknown, but b_0 varied within a narrow range (Voigt & Macdonald 1984).

(a) *Population equilibrium*

Simulations were carried out using one healthy pair of foxes per cell and no young as initial distribution levels.

First, for a constant survival rate ($\delta = 0$), there was a threshold value $b_{\min}(0)$ close to 1.02; if $b_0 < b_{\min}(0)$ the population goes extinct and if $b_0 > b_{\min}(0)$ the population follows a Malthusian growth pattern. For different birth rates, the corresponding Malthusian parameters have been determined (table 2). As in the one-dimensional model (Suppo 1996), for a birth rate $b_0 = 2.9$ this parameter is 0.016.

Second, with a density-dependent survival rate ($\delta > 0$), after a transient period a maximum yearly periodic distribution of individuals will be observed for $b_0 > b_{\min}(\delta)$, while the population will become extinct for $b_0 < b_{\min}(\delta)$. Eventually, the maximum number of foxes will be achieved on 1 April and can be determined with a suitably designed δ. Typically, a fox group on 1 April will be composed of one male, two fertile females and their litters, i.e. an average of 13 individuals for $b_0 = 2.5$ (Artois 1989). In the model this will occur when $\delta = 0.0025$ and $b_0 = 2.5$. For $\delta = 0.003$ the minimum threshold birth rate, $b_{\min}(0.003)$, needed to prevent the population from going extinct is close to 1.3. For birth rates varying up to $b_0 = 3.5$, the number of foxes per home range was determined for different values of δ (figure 3). A total of 13 foxes per home range can be obtained with different combinations of δ and b_0.

From a numerical point of view, we proceeded along two lines in order to get a prescribed maximum number of foxes on 1 April. Assuming this maximum to be 13 and $b_0 = 2.5$, we first put a pair of healthy adults and no young in each cell and ran the program until a yearly periodic distribution of individuals was achieved. Using a dichotomy method we estimated $\delta = 0.0025$, the average transient time being 12 years. We next modified the initial distribution of healthy foxes and re-ran the program with $\delta = 0.0025$ until a yearly periodic distribution of individuals was achieved; we again found 13 to be the maximum number, with variable transient times.

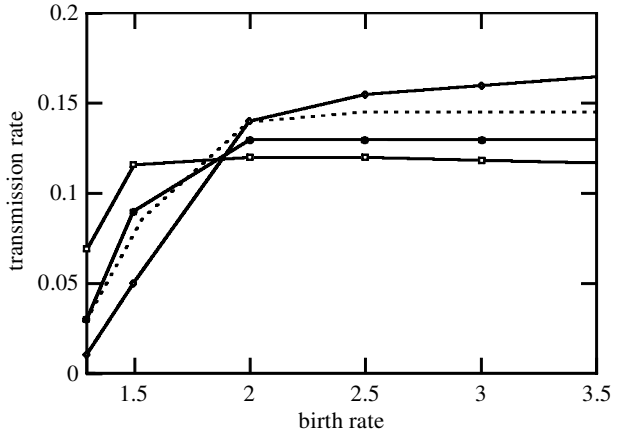

Figure 4. Maximum transmission rate $\beta_{\max}(b_0,\delta)$ for $\delta = 0.003$ (open diamonds), 0.002 (dashed line), 0.001 (asterisks) and 0 (open squares).

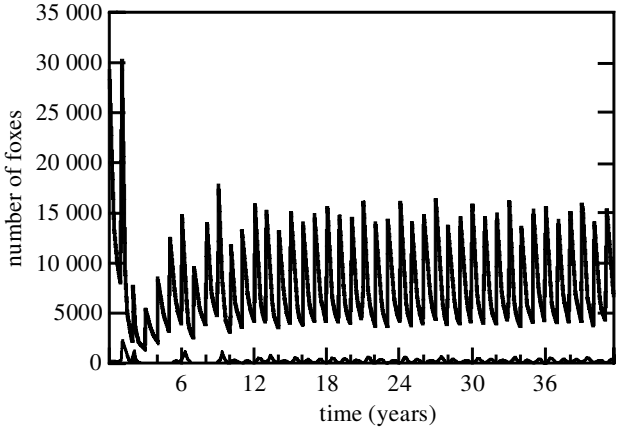

Figure 5. Dynamics of rabies for $b_0 = 2.5$, $\beta = 0.08$ and $\delta = 0.003$: healthy individuals (top curve) and infected individuals (bottom curve).

(b) *Rabies-endemic equilibrium*

In this section, we introduced a pair of exposed adult foxes in a single cell located at the centre of the domain and assumed that each cell contained one pair of healthy foxes.

Assuming a Malthusian growth trend ($\delta = 0$), we first determined the set of pairs (β,b_0) needed to yield an endemic state. Results show that for each fixed birth rate, b_0, rabies will not remain if the transmission rate is larger than a maximum threshold $\beta_{\max}(b_0,0)$ (figure 4).

Furthermore, the transmission rate must also be larger than a more or less constant value to further an endemic state, $\beta_{\min}(b_0,0) = 0.04$. For $b_0 = 2.5$ and $\beta = 0.08$ numerical simulations show similar results to those obtained in the

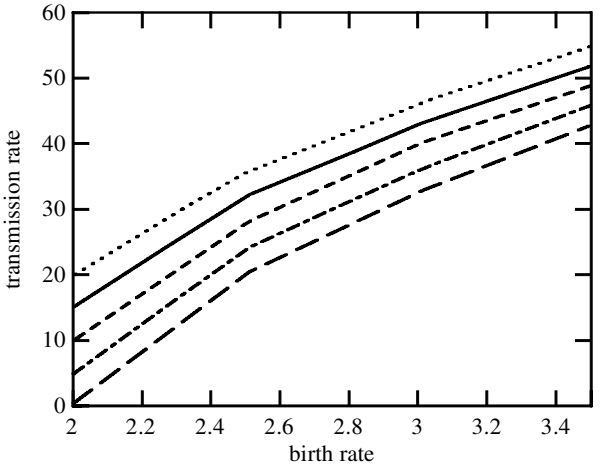

Figure 6. Sterilization rate required to eradicate rabies for $\delta = 0.003$, transmission rates $\beta = 0.06$ (top curve), 0.08, 0.10, 0.12 and 0.14 (bottom curve); variable birth rates.

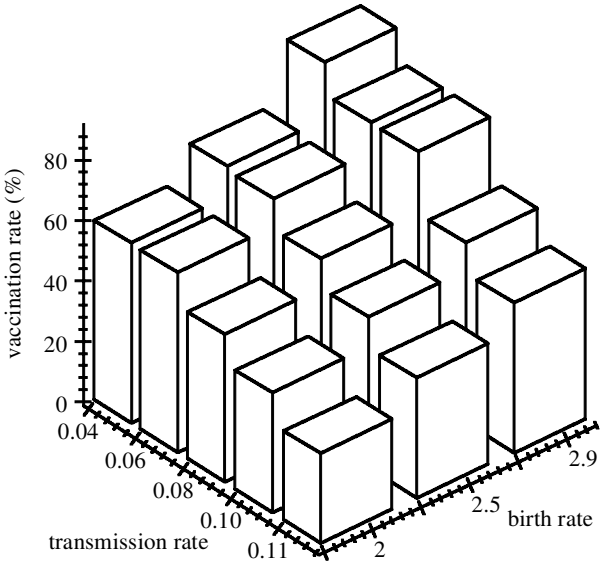

Figure 7. Vaccination rate required to eradicate rabies for different pairs of β and b_0 for $\delta = 0$.

one-dimensional model (Suppo 1996): between two waves, the population resumes a Malthusian growth trend as in a disease-free situation; during the first ten years the occurrence of four successive waves with high prevalence of infection was observed; during the next 30 years, the number of successive waves of rabies increased, the growth of the healthy population was regulated by rabies and a periodic endemic state emerged (figure 5).

For a logistic situation, the same pairs (β,b_0) were determined for different values of δ (see figure 4). For $b_0 \geqslant 1.7$ rabies could be sustained with higher transmission rates when $\delta > 0$ than when $\delta = 0$; this threshold $\beta_{\max}(b_0,\delta)$ increased with δ and b_0. Again, the transmission rate had a minimum threshold $\beta_{\min}(b_0,\delta)$ to further an endemic state, but the latter was strictly larger than 0.04, increased with δ and decreased with b_0. Finally, there existed an optimum transmission rate $\beta_{\text{opt}}(b_0,\delta)$, $\beta_{\min}(\delta) < \beta_{\text{opt}}(b_0,\delta) < \beta_{\max}(\delta)$, at which the prevalence was maximal (see § 3(d)). These results show that for a given transmission rate, if the birth rate decreased below 1.7, an endemic state could not be obtained and rabies disappeared. Thus, depending on the size of the birth rate, a sterilization method could decrease this rate and lead to eradication of rabies. In addition, for a birth rate $b_0 = 2.5$ and a transmission rate $\beta = 0.07$ rabies waves will occur every six or seven years.

If δ is close to zero, results would be similar to the Malthusian growth model.

(c) *Efficiency of fertility control*

The efficiency of sterilization programmes could be deduced from the previous computations. We can draw different conclusions from Malthusian and logistic growth trends.

First, we will consider a Malthusian growth trend (see figure 4). For a fixed transmission rate lying between 0.04 and 0.12, in order to eradicate rabies an initial birth rate in the range 2–3.5 must be decreased to a value close to $b_{\min}(0)$. Thus, it would be difficult to employ a sterilization method alone because a healthy population would go extinct before rabies was eradicated.

Let us also consider a logistic growth with $\delta > 0$, (see figure 4). For a transmission rate lying between $\beta_{\min}(b_0,\delta)$ and $\beta_{\max}(b_0,\delta)$, for rabies to disappear an initial birth rate in the range 2–3.5 must be decreased to a minimum value $b_{\text{opt}}(\delta,\beta) > b_{\min}(\delta)$. Consequently, an efficient sterilization rate could be determined for different birth rates and a fixed transmission rate (figure 6): for $\delta = 0.0025$, $b_0 = 2.5$ and $\beta = 0.08$ a sterilization rate close to 35% is required.

(d) *Efficiency of vaccination against rabies*

In our computations, vaccination programmes would begin after three years, corresponding to the control of an unexpected outbreak of rabies spreading quickly across the spatial domain. The vaccination rate was considered as efficient when rabies was totally eradicated; numerically this means the total number infected in the whole domain equal to zero for at least 20 years.

First, for a Malthusian growth trend, a minimum efficient vaccination rate was determined for various pairs (β,b_0) in order to eradicate rabies (figure 7). This minimum rate was larger in high-density populations and decreased when β increased. For $b_0 = 2.5$ and $\beta = 0.06$, simulations gave the minimum efficient rate of vaccination necessary to eradicate rabies as 70%, which is close to the upper limit achieved in the field during actual vaccination campaigns (Aubert 1995). In other words, both numerical simulation and field findings show that there is a density at which vaccination fails to eradicate rabies.

Second, for a logistic growth trend, the same computations were carried out to emphasize the difference for lower-density populations; see figure 8 for $\delta = 0.003$. According to the results, in order for a vaccination to be efficient the rate must be higher for high birth rates (corresponding to a larger population), with maximum values at the optimum transmission rate $\beta_{\text{opt}}(b_0,\delta)$.

It is worth noting that in both cases (logistic and Malthusian) the dynamics of rabies was modified by low vaccination rates. For a vaccination rate between 20 and 30% (figure 9), the first wave of rabies was delayed, but afterward the number of successive waves increased and a new one could occur every year with the same prevalence. For a rate lower than, and close to, the efficient

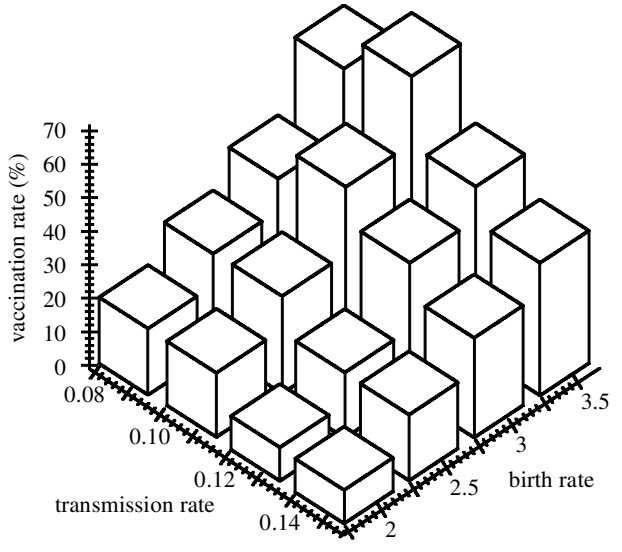

Figure 8. Vaccination rate required to eradicate rabies for different pairs of β and b_0 for $\delta = 0.003$.

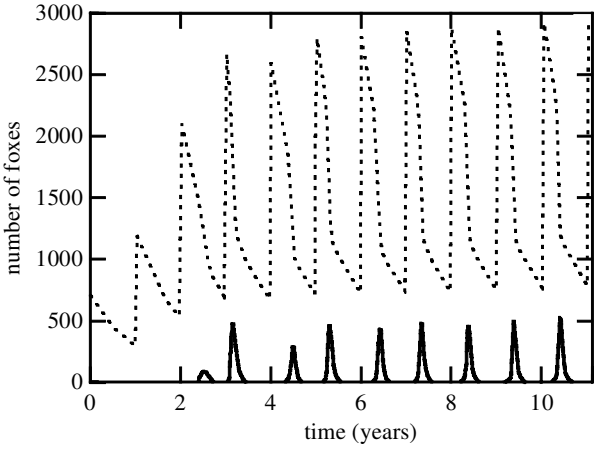

Figure 9. Dynamics of rabies for a vaccination rate close to 25%: healthy individuals (dotted line) and infected individuals (solid line).

rate, the first wave appeared later but the prevalence of following waves increased continuously.

Now we come to the key point of our analysis. For some pairs of b_0 and β, the vaccination rate needed to eradicate rabies had to be larger than 70%, which is difficult to achieve in the field (Breitenmoser *et al.* 1995; Vuillaume *et al.* 1997). In these cases contraception is required to improve the efficiency of anti-rabies vaccination.

(e) *Vaccination and fertility control combined*

For a Malthusian growth trend, we saw that contraception alone could lead to extinction of foxes (see §3(c)). A combination of both methods could be efficient if birth rates were decreased to a value requiring a lower vaccination rate. It is straightforward to observe from figure 7 that coupling vaccination and sterilization could be successful. Thus, we could fix several vaccination rates less than or equal to that needed to be successful when vaccination alone was used. We could then deduce the minimum sterilization rate required to eradicate rabies. Figure 10 shows the required combinations for $\beta_{\min}(3,0) = 0.04$ and $\beta_{\max}(3,0) = 0.11$.

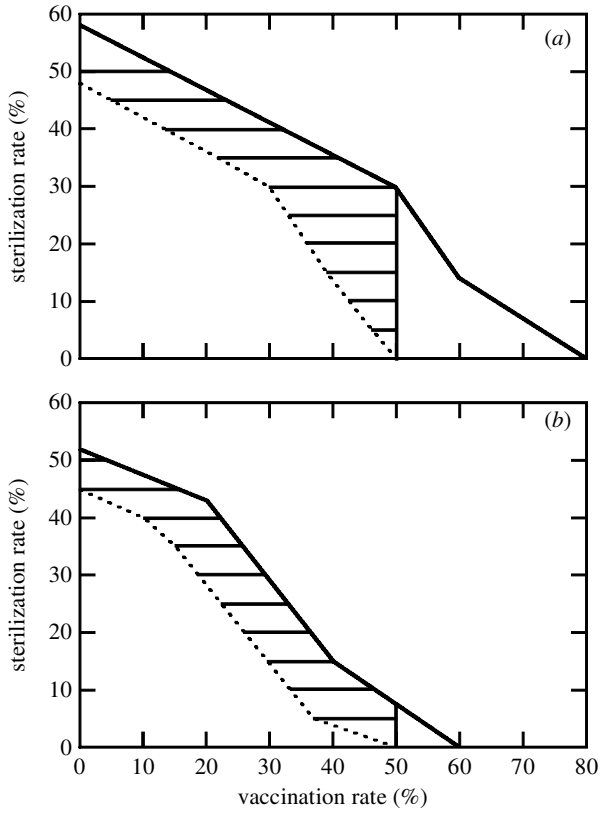

Figure 10. The shaded area contains combinations of vaccination and sterilization rates required, that could be achieved in the field, to eradicate rabies for (*a*) $\delta = 0$ and $\beta_{\min}(3,0) = 0.04$ (dashed line) $\leqslant \beta \leqslant 0.11 = \beta_{\max}(3,0)$ (solid line) and (*b*) $\delta = 0.003$ and $\beta_{\min}(3.5,0.003) = 0.08$ (dashed line) $\leqslant \beta \leqslant 0.14 = \beta_{\max}(3.5,0.003)$ (solid line).

For a logistic growth trend, we saw that a successful sterilization rate could be determined but would be difficult to obtain. A combination of both control methods is also beneficial (see figure 8). For $\delta = 0.003$, figure 10 shows the successful combinations of sterilization and vaccination for $\beta_{\min}(3.5,0.003) = 0.08$ and $\beta_{\max}(3.5,0.003) = 0.14$. We observed a linear relationship between the vaccination and sterilization rates; linear regression yields a slope equal to -0.76 for $\beta_{\min}(b_0,0.003)$.

4. DISCUSSION

Over time, more than 15 models have been devoted to fox rabies (reviews in Barlow (1995) and Pech & Hone (1992)). The main value of the one herein presented lies in the use of recent and actual data from fox baiting in France (Aubert 1995). Additionally, the use of contraception to manage rabies in fox populations is considered (Artois & Bradley 1995). As with many models of the same type, ours is oversimplified in several regards and some of the results obtained could be consequences of these oversimplified choices.

We have omitted differences between the dispersal modes of male and female animals. Also we do not take into account the effect of culling, because its efficiency has not been fully demonstrated on the European continent (Aubert 1994). Therefore, it was considered that, to a large extent, fox control by various methods constituted a part of the density-dependent mortality. Additionally,

density dependence in this study acted only on survival and not on reproduction. This is close to what has been observed in nature: a lack of variability in the fertility rate within a wide range of natural conditions suggests that fecundity is a stable demographic parameter in Europe. Finally, the dispersal mode used in this model enabled us to estimate the population size after yearling dispersal, but did not simulate a preferred settlement of young in less densely occupied areas. Knowledge about dispersal patterns that include this behaviour is currently so limited (see Lloyd 1980; Harris 1981; Macdonald & Bacon 1982; Trewhella *et al*. 1988; Allen & Sargeant 1993) that this simplification is worth keeping.

Obvious trends in fox demographic indices suggest a steady population increase over the long term (Müller 1995; Artois *et al*. 1997). Ecological reasons for this population increase remain unclear, but links with a decrease in human control seem the most likely explanation (Aubert 1994; Szemethy & Heltai 1997). These trends were simulated in this model through a Malthusian growth process obtained by a constant that ensured reproduction greater than mortality. Trends in the model are similar but not precisely adjusted to those observed under field conditions.

Socio-spatial adjustment with increasing density was not considered in this model. This could have an influence in a spatial model of rabies diffusion: in brief, foxes are regarded as 'contractors' (Kruuk & Macdonald 1984) maintaining the smallest economically defensible home range. Additional residents would be tolerated as long as sufficient resources are available (compatible with the resources dispersion hypothesis, see Carr & Macdonald (1986)). According to field observations, the number of adult individuals within a social group is nevertheless limited to four or five (constant territory size hypothesis (CTH) versus territory inheritance hypothesis (TIH), see Lindström *et al*. 1982; Von Schantz 1984; Lindström 1986). Few behavioural studies have been recently devoted to this aspect of the spatial behaviour of foxes. Therefore, the response to a decrease in mortality within a situation of stable accessibility to resources is unknown. The model herein presented accepts the unverified hypothesis that under these conditions the number of individuals within a social group could reach a limit transgressing the CTH–TIH hypotheses. Further field research is needed to clarify this aspect. Concerning the propagation of the virus, a uniform transmission rate was used; there is then no variation due to sex or age (dispersers could be less exposed than resident adults, see Artois & Aubert (1985)), and no variation in contact rate between foxes living within the same or in adjacent territories (see Artois & Aubert 1985).

Our model is considered in a constant environment, unlike that of Pech *et al*. (1997), who have studied the effect of environmental variability on the use of fertility control of foxes in arid Australia. No compensatory phenomena (Hone 1994) to contraception, such as an increase in the birth rate of non-sterilized females (Newsome 1995), an increase in the survival rate of foxes, or immigration (Seagle & Close 1996), were introduced in our model. Therefore, no side effects or retarded effects could be expected in our short-term analysis; with the purpose of this project being the fast control of an outbreak of rabies, long-term effects did not need to be considered.

In the conditions described by our model of an isolated host population, one observes that a stable endemic equilibrium emerges with rabies regulating the fox population in both demographic settings, i.e. logistic and Malthusian. This occurs when demographic and epidemiological parameters lie within a reasonably realistic range. Under our assumptions this stable equilibrium between the virus and the host requires a fast turn-over of the healthy foxes. Since survival of the population is assured by dispersal (October) and reproduction (April), it is understandable that for a small transmission rate the virus does not propagate at a sufficient speed to survive, while for a large transmission rate the mortality due to rabies cannot be compensated for in time. Still under our assumptions, it follows that the propagation of the epidemic disease is not very sensitive to dispersal, while being more sensitive to birth rate. This should moderate biological considerations that could be drawn from our model. Nevertheless, provided that these simplifications can be accepted, the model suggests that sterilization turns out to be a strong complement for controlling fox rabies. Additionally, our model suggests that density dependence smoothes out fluctuations at equilibrium between the host and the virus. In contrast with intuitive predictions, the successful rate of rabies eradication is higher when the host population is not regulated, i.e. Malthusian growth.

Nevertheless, for a fox population experiencing a Malthusian growth curve, vaccination alone would be inefficient to eradicate rabies, as expected, so sterilization turns out to be specifically helpful here.

Finally, as an alternative approach we compared our results to a deterministic and time-continuous model, given in an electronic appendix (http://durandal.mass.u-bordeaux2.fr/-naulin/appendix/appendix.html), based on a system of ordinary differential equations such as that used in Anderson *et al*. (1981) and Barlow (1996). As a first difference this continuous model does not predict self-eradication of rabies when the transmission rate is large. Also, for a Malthusian population growth trend, the vaccination efficiency cannot be predicted by the model and depends on the parameters defining the initial state. Nevertheless, similar conclusions concerning sterilization can be drawn from both continuous and discrete models. Discrete-time modelling appears, then, to be more appropriate for our purpose. Predictions obtained from both models are encouraging in considering immunocontraception as a possible method of controlling a re-emerging outbreak of rabies in highly dense fox populations. Nevertheless, additional biological hypotheses that need to be taken into account in further studies include fox culling considered as a non-density-dependent mortality factor, changes in spacing strategies when density increases, and the influence of dispersal in the recovery of healthy populations.

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